AMENDMENTS TO THE CLAIMS:

Claims 1-4 (Withdrawn)

Claims 5-12 (Canceled)

Claims 13-16 (Withdrawn)

Claims 17-30 (Canceled)

Claims 31-33 (Withdrawn)

34. (New) A transgenic mouse whose genome comprises a disruption in endogenous mouse anaphylatoxin C3a receptor gene, wherein where the disruption is homozygous and the mouse is male, the transgenic mouse lacks production of functional anaphylatoxin C3a receptor and exhibits, relative to a wild-type mouse, reduced thymus weight, reduced thymus size, reduced thymus to body weight ratio, increased susceptibility to seizure or a stimulus processing deficit, and wherein where the disruption is homozygous and the mouse is female, the transgenic mouse lacks production of functional anaphylatoxin C3a receptor and exhibits, relative to a wild-type mouse, increased susceptibility to seizure or a stimulus processing deficit.

35. (New) The transgenic mouse of claim 34, wherein the increased susceptibility to seizure is characterized by a lower dose of metrazol required to reach characteristic stages of seizure, relative to a wild-type mouse.

36. (New) The transgenic mouse of claim 34, wherein the stimulus processing deficit is characterized by a decrease in prepulse inhibition, relative to a wild-type mouse.

37. A cell or tissue obtained from the transgenic mouse of claim 34.

38. A transgenic mouse comprising a heterozygous disruption in endogenous mouse anaphylatoxin C3a receptor gene, wherein the disruption in a homozygous state inhibits production of functional anaphylatoxin C3a receptor resulting in a male transgenic mouse exhibiting, relative to a wild-type mouse, reduced thymus weight, reduced thymus size, reduced thymus to body weight ratio, increased susceptibility to seizure or a stimulus processing deficit or a female transgenic mouse exhibiting, relative to a wild-type mouse, increased susceptibility to seizure or a stimulus processing deficit.

39. (New) The transgenic mouse of claim 38, wherein the increased susceptibility to seizure is characterized by a lower dose of metrazol required to reach characteristic stages of seizure, relative to a wild-type mouse.

40. (New) The transgenic mouse of claim 38, wherein the stimulus processing deficit is characterized by a decrease in prepulse inhibition, relative to a wild-type mouse.

41. A method of producing a transgenic mouse comprising a disruption in endogenous mouse anaphylatoxin C3a receptor gene, the method comprising:

(a) introducing a targeting construct capable of disrupting endogenous mouse anaphylatoxin C3a receptor gene into a mouse embryonic stem cell;

(b) introducing the mouse embryonic stem cell into a blastocyst;

(c) implanting the blastocyst into a pseudopregnant mouse, wherein the pseudopregnant mouse gives birth to a chimeric mouse; and

(d) breeding the chimeric mouse to produce the transgenic mouse comprising the disruption in endogenous mouse anaphylatoxin C3a receptor gene;

wherein where the disruption is homozygous and the mouse is male, the transgenic mouse lacks production of functional anaphylatoxin C3a receptor and exhibits, relative to a wild-type mouse, reduced thymus weight, reduced thymus size, reduced thymus to body weight ratio, increased susceptibility to seizure or a stimulus processing deficit, and wherein where the disruption is homozygous and the mouse is female, the transgenic mouse lacks production of functional anaphylatoxin C3a receptor and exhibits, relative to a wild-type mouse, increased susceptibility to seizure or a stimulus processing deficit.

42. The transgenic mouse produced by the method of claim 41.